Ultrasound in Pulmonary Embolism

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KEYWORDS

• Pulmonary embolism • Venous thromboembolism • Right ventricular failure
• Point-of-care ultrasound • Echocardiography

KEY POINTS

- Acute pulmonary embolism (PE) has a high morbidity and mortality, requiring prompt diagnosis and treatment.
- Most singular sonographic findings suggestive of acute PE have low sensitivity but very high specificity.
- High pre-test probability, hemodynamic instability, and a multi-organ ultrasound approach improves ultrasound test performance characteristics.
- Ultrasound plays a crucial role in the triaging, diagnosis, risk-stratification, and prognostication of acute PE, with a multi-organ comprehensive approach.

Video content accompanies this article at http://www.criticalcare.theclinics.com.

INTRODUCTION

Pulmonary embolus (PE) is an obstruction of the pulmonary arterial circulation by a thrombus originating elsewhere in the body. PE is the third leading cause of death in the United States and Western Europe.[1] In the United States alone, PE is responsible for approximately 300,000 deaths per year.[2,3] Many cases of sudden cardiac death are also thought to be secondary to PE, so the actual mortality attributable to PE is difficult to estimate.[4] The overall 90-day mortality for all risk groups of PE is 16% to 18%, which has remained unchanged over the last 3 decades. However, the mortality in PE is dependent on its hemodynamic sequelae, with intermediate-

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Abbreviations	
A4C	apical four-chamber
CO	cardiac output
DVT	deep vein thrombosis
E_a	arterial elastance
E_{es}	end-systolic elastance
HR	hazard ratio
LV	left ventricle
LVOT	Left ventricleoutflow tract
NPV	negative predictive value
OR	odds ratio
PAP	pulmonary artery pressure
PASP	pulmonary artery systolic pressure
PE	pulmonary embolus
PI	pulmonary infarct
POCUS	point-of-care ultrasound
PSAX	parasternal short axis
PVR	pulmonary vascular resistance
RV	right ventricle
RV FAC	right ventricular fractional area change
RVOT	right ventricle outflow tract
RV S′	tricuspid annular peak systolic velocity
RVEDV	right ventricle end diastolic volume
SV	stroke volume
TAPSE	tricuspid annular plane systolic excursion
TRPG	tricuspid regurgitation pressure gradient
VTE	venous thromboembolism
CIT	clot-in-transit
RVLS	right ventricle longitudinal strain
BLUE	bedside lung ultrasound in emergency

risk PE carrying a 90-day mortality of 12% to 15%, which increases to 40% to 50% for the high-risk PE, and further up to 70% once cardiac arrest occurs.[5–7] Given this high mortality, prompt diagnosis, accurate risk stratification, and early institution of appropriate treatment are crucial.

In primary or idiopathic PE, multiple risk factors are implicated including diabetes, metabolic syndrome, tobacco use, obesity, and hypertension.[1] In hospitalized patients, acute illness, infection, and immobilization increase the risk of deep vein thrombosis (DVT) and PE, with venous thromboembolism (VTE) developing in up to 30% of ICU patients despite universal use of pharmacologic and mechanical thromboprophylaxis.[8] Anticoagulation is the standard of care for low-risk PE, with advanced therapies (ie, catheter directed thrombolysis, aspiration, or surgical thrombectomy) reserved for selected intermediate-high and high-risk patients. A key in the management of PE is prompt definite diagnosis of presence of PE and risk stratification, so appropriate intervention can be instituted. Point-of-care ultrasound (POCUS) is critical in triaging and managing patients with acute PE. POCUS provides both accurate measurements, cardiac sequala of PE, and possesses the versatility to obtain collateral data, such as evaluating for pulmonary infarcts (PI) and DVT. This article provides an in-depth review of the pathophysiology of PE, role of multimodal POCUS in diagnosis and risk stratification of PE.

Pathophysiology of Pulmonary Embolism

Virchow's triad encompasses the risk factors that contribute to thrombosis—stasis of blood flow, vascular endothelial damage, and hypercoagulability, which can be

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inherited or acquired (eg, surgery, trauma, immobilization, medications, active cancer). All patients who experience PE generally have at least one risk factor.[9] Most PEs originate as a thrombus in the deep veins of the lower extremities, most frequently femoropopliteal veins, the iliac veins, and the calf veins. Lower extremity central DVTs cause PE in 15% to 32% of the cases.[10] PEs can also arise from DVT in the renal and upper extremity veins, although embolization from these veins accounts for only 6% of PE.[11,12] These emboli detach from their origin site and enter pulmonary circulation via the venous system obstructing blood flow. The symptoms range from being asymptomatic to having chest pain, shortness of breath, hypoxemia, cardiovascular collapse, and in extreme cases, sudden cardiac death.[13]

Pathophysiology of right ventricular failure in pulmonary embolism

The hemodynamic consequences observed in PE are proportional to the impact of the clot burden on right ventricle (RV) physiology.[6] The thin-walled RV is preload tolerant but very sensitive to sudden increases in afterload.[14,15] When RV afterload or pulmonary vascular resistance (PVR) abruptly increases with the increase in pulmonary artery pressure (PAP), the stroke volume of the RV drops precipitously in contrast, to left ventricle (LV) as shown in **Fig. 1**.[16] Of note, RV dysfunction with PE increases mortality, even in the absence of apparent hemodynamic compromise.[17,18] In addition, RV dysfunction in PE also has prognostic implications beyond the acute setting, as persistent RV dysfunction at discharge increases the risk of recurrent VTE and carries a mortality 4 times higher in contrast to those with resolution of RV dysfunction.[19]

To understand the insults to the RV in acute PE that initiate this *death spiral*, two important physiologic concepts are important to understand: ventricular interdependence and ventriculoarterial coupling.[20,21]

Ventriculoarterial coupling. Ventriculoarterial coupling refers to the efficiency of mechanical energy transfer from the ventricle to the vasculature. In the case of PE, the ventriculoarterial coupling is from a thin-walled RV to a low-pressure pulmonary vascular tree.[20] For efficient energy dynamics and blood flow, the RV needs to optimize

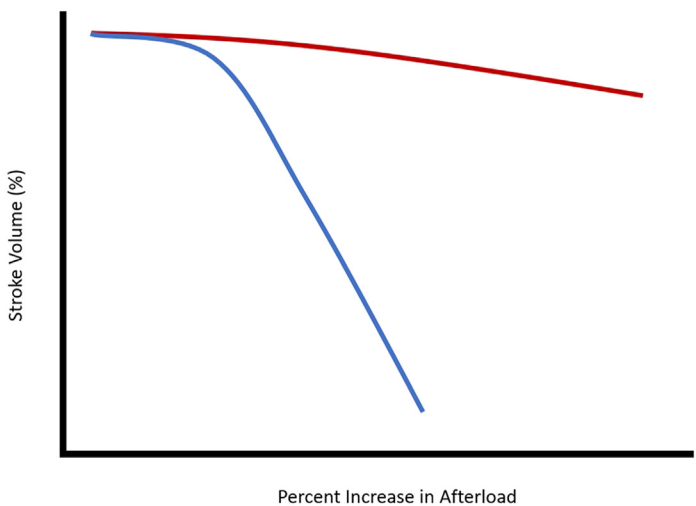

Fig. 1. A graphical depiction of the relationship between stroke volume and afterload, highlighting the stark differences in the behavior of the left ventricle (red) and the right ventricle (blue).[16]

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contractility to adapt to changes in afterload. This homeometric adaptation of RV contractility happens within minutes of increased afterload and is assessed by the relationship of ventricular end-systolic elastance (E_{es}) to pulmonary arterial elastance (E_a), or E_{es}/E_a ratio. For the right heart, a normal E_{es}/E_a is 1.5 to 2.0.[20,22] If this homeometric (ie, contractility) adaptation is insufficient, the RV undergoes heterometric adaptation (ie, dilatation), with increased RV end diastolic volume (RVEDV) to maintain stroke volume. In patients with chronic pulmonary hypertension, mortality increases once this optimal E_{es}/E_a ratio cannot be maintained, and uncoupling develops.[23] In contrast to chronic pulmonary hypertension where right ventricular-pulmonary artery (RV-PA) uncoupling develops slowly after exhaustion of compensatory mechanisms, RV-PA uncoupling happens abruptly in acute PE, see **Fig. 2**.

Ventricular interdependence. The differences in ventricular chamber pressure and constraints of the pericardium determine the septal kinetics. In normal health, LV chamber pressure is higher than the RV, resulting in a convex septum bowing toward the lower pressure RV, but reverses and bows toward the LV when the RV pressure exceeds the LV. In setting of acute PE, the distressed and dilated RV continues to contract after LV contraction has ended, resulting in a momentary increase in RV pressure.[24]

This change in configuration of the septum is visualized as the flattening of the interventricular septum to give the LV a *D-shaped* appearance, as seen in **Fig. 3**. This progressive LV obliteration diminishes LV preload, cardiac output, and mean arterial pressure, further undermining coronary perfusion to the RV with fatal consequences.[25]

Diagnosis of Pulmonary Embolism by Ultrasound

PE has a wide spectrum of clinical presentations, and in most cases, the presenting symptoms are nonspecific and hence require a high degree of suspicion on the part of the clinician. Multiple PE diagnostic scoring systems exist including the Wells criteria and the Geneva score to determine the pre-test probability of PE.[26,27] Lung, cardiac, and deep venous thrombosis (DVT) ultrasound are excellent tools to augment the performance characteristics of the Wells scoring system, especially increasing the sensitivity when it is paired with a negative D-dimer.[28]

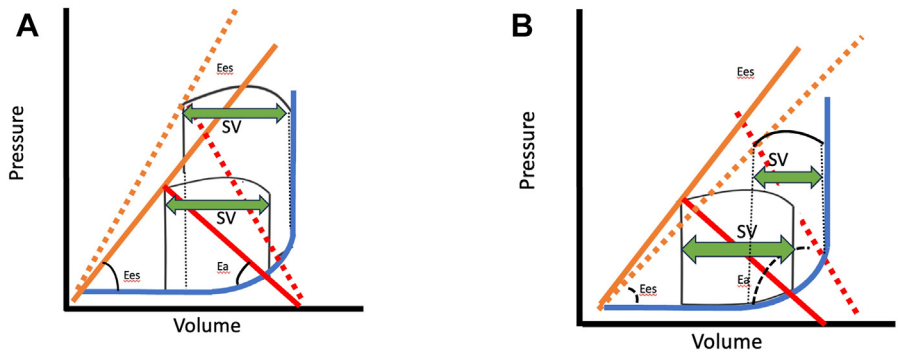

Fig. 2. (*A*) RV PA loop with normal E_{es}/E_a ratios and stroke volume. Superimposed RV, PA loop in pulmonary hypertension with preserved E_{es}/E_a ratios (*dashed slopes*), with increased end-systolic and end-diastolic pressures and unchanged stroke volume (*green arrows*). (*B*) RV PA loop with normal E_{es}/E_a ratios and stroke volume. Superimposed RV PA loop in acute pulmonary embolism showing decreased E_{es}/E_a ratio (*dashed slopes*), increased afterload and decreased stroke volume (compare *green arrows*).

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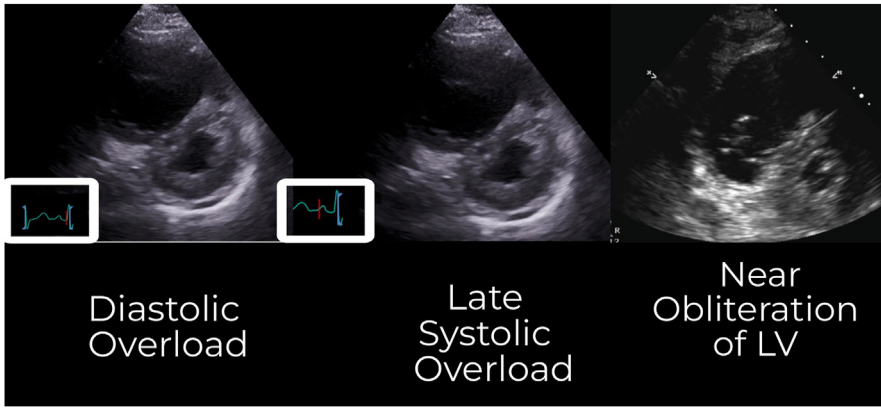

Fig. 3. Echocardiogram in a patient with acute PE showing septal flattening and "D-shaped" septum in the parasternal short-axis view. Combined volume & pressure overload seen with near obliteration of the LV at the end of systole.

Venous ultrasound

POCUS DVT examination has been proven to be highly sensitive and specific in the emergency department and intensive care units.[29–31] The POCUS DVT examination is usually done at 2 areas: the inguinal region and the popliteal fossa. Using a high-frequency ultrasound probe in short-axis, the visualized veins are physically compressed by applying downward pressure on the ultrasound probe and observing for complete compressibility of the common femoral vein, the saphenofemoral junction, and bifurcation of the femoral and deep femoral vein. The popliteal vein is also visualized in the popliteal fossa and compressed to the confluence of the distal calf veins.[32] In a systematic review of 16 studies, the sensitivity and specificity of 2-zones and 3-zones POCUS DVT examination were over 90%.[33] However, Adhikari and colleagues showed that 6% patients with suspected DVT have isolated femoral vein thrombi, which could be missed with the two-point technique.[34] Extended compression ultrasound, which evaluates the entire femoral vein from the femoral/deep femoral vein bifurcation to the adductor canal, is recommended over other protocols.[35] Other advanced techniques like color Doppler or spectral Doppler can also be used to determine the degree of occlusion. A positive examination entails a visualizing echogenic thrombus or a noncompressible vein, as seen in **Fig. 4**, and carries a high specificity (96%) but low sensitivity (41%).[36]

Lung ultrasound

Lung ultrasound is a remarkable tool in assessing patients presenting with undifferentiated dyspnea. The lung ultrasound findings in PE can range from normal lung parenchyma with A-line pattern to finding of pulmonary infarct (PI) and pleural effusion.[37] The bedside lung ultrasound in emergency (BLUE) protocol uses a combination of A-line pattern with detection of venous clot to confirm the diagnosis of PE.[38] Lung ultrasound findings of PI include the classic triangular wedge-shaped or round subpleural consolidation pleural-based lesion, as seen in **Fig. 5**.[39]

Other specific PI findings in CT, such as "bubbly consolidation" have their equivalent counterpart by lung ultrasound, with triangular hypoechoic consolidation, sharp margins, and a central roundish hyperechoic area.[40] More than 2 decades ago, high specificity (>90%) for these lung ultrasound findings was reported in detection of PE albeit, with low sensitivity.[41,42] The sensitivity improves to 82% for detecting PI if

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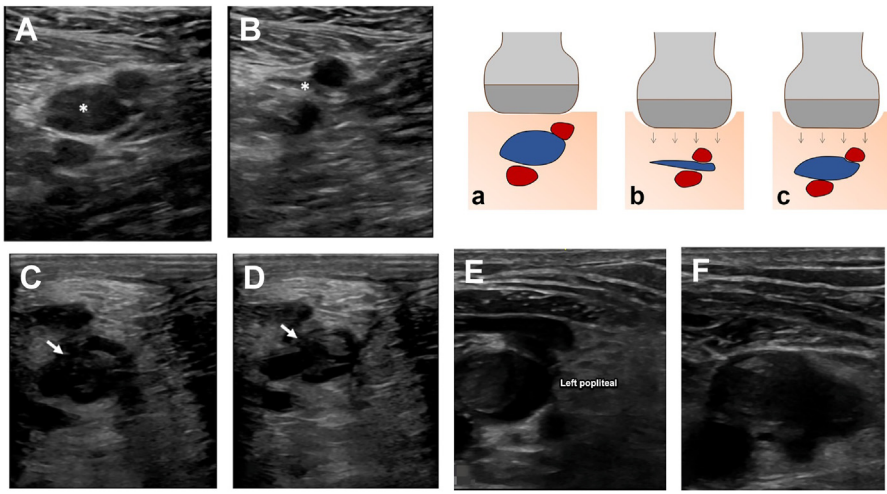

Fig. 4. Images of a patient with an acute DVT. (*A*) The common femoral vein marked by '*', (*B*) the same anatomic location under compression, demonstrating complete obliteration of the common femoral vein. (*C*) The contralateral extremity femoral vein without compression, (*D*) the same anatomic location under compression with the arrow indicating lack of compressibility. (*E*) showing echogenic mass in left popliteal vein and lack of compressibility (*F*) consistent with left popliteal DVT. Illustration (*a*) illustration of vessel (*b*) showing complete venous compression (*c*) lack of venous compression.

patient presents with pleuritic chest pain. A meta-analysis of 10 studies found lung ultrasound to have a mean sensitivity of 87.0% and specificity of 81.8%. The authors calculated sensitivity to be 89%, when PE was located at main, lobar, or segmental pulmonary artery branches, which dropped to 21% with distal PE.[43]

While lung ultrasound can identify PI, suggesting the presence of PE, it is also effective in confirming or excluding alternative causes of clinical symptoms that overlap with PE, such as pleural effusions, pulmonary edema, and pneumonia.[44,45]

Cardiac ultrasound

Echocardiographic assessment includes evaluation of RV dimension, systolic function, flow or stroke volume surrogates, filling pressures, physiologic interactions of

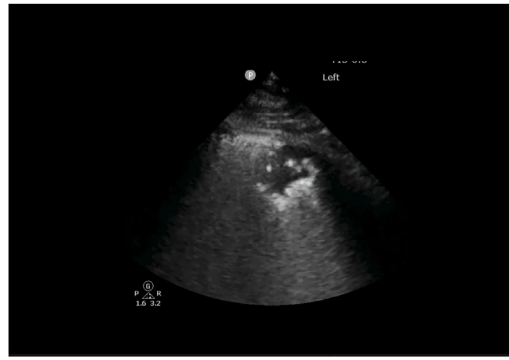

Fig. 5. Pulmonary Infarct. Rounded hypoechoic pleural based lesion with irregular margins.

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the left and right ventricles, and ventriculoarterial coupling. Echocardiography has limited diagnostic utility given the low sensitivity of echocardiographic findings in PE in the absence of right ventricular dysfunction. In a cohort of 511 patients, all hemodynamic unstable PE patients had some echocardiographic signs of RV dysfunction, which reduced to 65% in hemodynamically stable PE.[46] According to the European Society of Cardiology guidelines, the presence of RV dysfunction on echocardiography suffices for diagnosing and treating PE in suspected patients who are too unstable to undergo a CT pulmonary angiogram.[47] In other words, the performance characteristics of echocardiographic findings in PE are dependent on the pre-test probability of PE and clinical conditions,[48] with high sensitivity and specificity in high suspicion for PE and shock, where normal RV size and function virtually eliminates hemodynamically significant PE, and the presence of acute RV dysfunction is highly suggestive of PE as the cause of shock.[46]

Right ventricular dimensions. Echocardiographic evaluation of RV dimensions is cumbersome due to its crescent nontraditional geometric shape.[49] It is important to assess the maximal diameter of the RV, which is recommended in an RV-focused view, to avoid underestimating the RV's maximal basal and midlevel diameter.[50] Even though RV dilation is considered when RV basal diameter is over 41 mm (**Fig. 6**) in an RV-focused view, most studies done in PE classify RV dilation, when RV end-diastolic diameter (RVEDD) is more than 27 mm to 30 mm in an apical four-chamber (A4C).[48,51,52] In one study, RVEDD more than 27 mm along with either TR velocity more than 2.7 m/sec or RV hypokinesis had a sensitivity of 56% (95% confidence interval [CI]: 40% to 71%) and specificity of 90% (95% CI: 80% to 96%) in diagnosing PE.[48] Combining vital signs improves the accuracy of echocardiography in identifying PE.[53] RV/LV ratio more than 0.7 to 0.9 has also been evaluated in multiple studies to diagnose PE with sensitivity ranging from 50% to 66% and specificity ranging from 77% to 98% based on the cutoff of RV:LV ratios applied.[51,54] In a meta-analysis, RVEDD had the highest sensitivity of all signs (83%), especially in physician-performed studies.[55]

In the setting of chronic pulmonary hypertension, the RV myocardial wall thickness increases to improve maximal contractility and maintain ventriculoarterial coupling.[23] This is assessed using echocardiography by measuring RV wall thickness in a subcostal view. An RV wall thickness more than 5 mm is highly indicative of compensatory changes to chronically elevated RV afterload. However, one must be careful, as PE can cause acute-on-chronic RV decompensation that may be challenging to identify.[50] See **Table 1** for RV wall thickness and chamber size assessment.

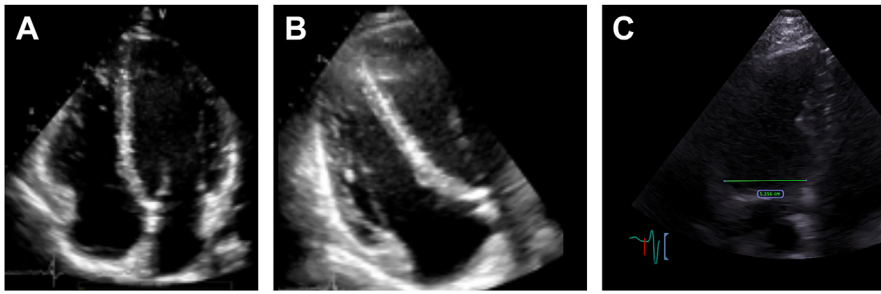

Fig. 6. The RV-focused view on transthoracic echo. The image on the left (*A*) shows an apical 4 chamber view and the image on the right (*B*) shows the apical RV-focused view with clear identification of the RV myocardium and free wall. (*C*) shows severe RV enlargement at the RV base in an RV modified A4C view.

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Table 1
Venous, lung, and echocardiographic findings in pulmonary embolism with their respective sensitivity, specificity, positive and negative likelihood ratios for diagnosing pulmonary embolism as previously reported[36,43,55,60,71,106]

Ultrasound Finding	Examination	Interpretation	Sens.	Spec.	+LR	-LR
Noncompressible vein	Femoral & Popliteal vein	Thrombus present	0.41	0.96	11.9	0.6
Pleural-based triangular consolidations	Lung ultrasound	Pulmonary infarct	0.85	0.82	5.6	0.78
RV/LV ratio >0.9	A4C	RV dilatation	0.55	0.86	3.9	0.5
RVEDD >30 mm	RV Focused	RV dilatation	0.80	0.80	4.0	0.25
Paradoxic septal motion	PSAX	Pathologic ventricular interdependence	0.26	0.95	5.2	0.78
60/60 Sign	A4C & PSAX PV valve	High pulmonary artery afterload	0.24	0.84	1.5	0.9
Early systolic notching of pulmonary arterial flow	PSAX PV or RV outflow view	High pulmonary artery afterload	0.92	0.99	92	0.08
RV hypokinesia	A4C or RV focused	Decreased RV systolic function	0.38	0.91	4.2	0.68
Reduced TAPSE	A4C or RV focused	Decreased RV systolic function	0.64	0.61	1.64	0.59
Clot-in-transit	Multiple views	Imminent PE	0.05	0.99	5	0.9
Multiorgan: LUS + venous US + echocardiography	Multiorgan POCUS	Multiple VTE manifestations	0.90	0.86	6.5	0.12

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Paradoxic septal motion. Echocardiography allows further evaluation of septal kinetics during the cardiac cycle (ie, pressure vs volume overload or both). One parameter that can be used to evaluate septal kinetics and differentiate pressure vs volume overload state is the eccentricity index.[56] The eccentricity index is the ratio of the minor (D2) and major axis (D1) diameters of the left ventricle in a parasternal short axis (PSAX) view at the papillary muscles level. A normal left ventricle is circular, so the ratio is close to 1. In patients with pressure overload, the index (D2/D1) is greater than 1 at the end of systole and equal to 1 at the end of diastole. In patients with volume overload (ie, atrial septal defect or tricuspid regurgitation without pulmonary hypertension), the eccentricity index is greater than 1 at the end-diastole, but equal to 1 at the end of the systole. In patients with acute PE, concomitant pressure and volume overload is often seen, as seen in **Fig. 7**.

In a prospective observational study, all patients with confirmed PE had PSM.[54] Another prospective observational ED study also showed a high specificity of 97% for PSM, but a sensitivity of only 19% for patients with suspected PE.[57] In a larger retrospective cohort of patients with confirmed PE, 18% of low-risk PE patients had PSM vs. 69% in those classified as high-risk PE.[46]

Pulmonary artery pressure

60/60 sign The 60/60 sign refers to pulmonary artery acceleration time of less than 60 ms with concomitant tricuspid regurgitation pressure gradient (TRPG) less than 60 mm Hg. Values of TRPG exceeding 60 mm Hg align with underlying chronic pulmonary hypertension (PH), and hence the value of the "60/60 sign" in acute PE without preexisting PH, **Fig. 8**. The specificity of the 60/60 sign has been reported to be as high as 98%, but with poor sensitivity to diganos PE.[58,59] The sensitivity was however, found to be higher (51%) in hemodynamically significant PE compared to subsegmental PE (7%).[60]

Early Systolic Notching The development of PH changes the "dome" shaped pulmonary arterial waveform to different patterns (most notably the early or midsystolic notching characteristics) as pulmonary vascular resistance rises causing transient decrease in velocity during the pulmonary arterial systolic flow cycle, see **Fig. 9**. Early systolic notching (ESN) defined as notching occurring within the initial 50% of RV systolic ejection period is a marker of elevated PAP and correlates with thrombus

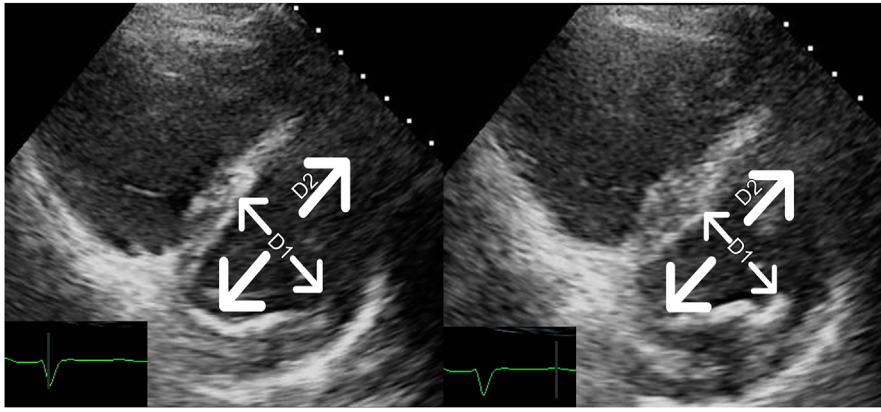

Fig. 7. Eccentricity Index >1 in diastole (*left*) and in systole (*right*) consistent with combined volume and pressure overload.

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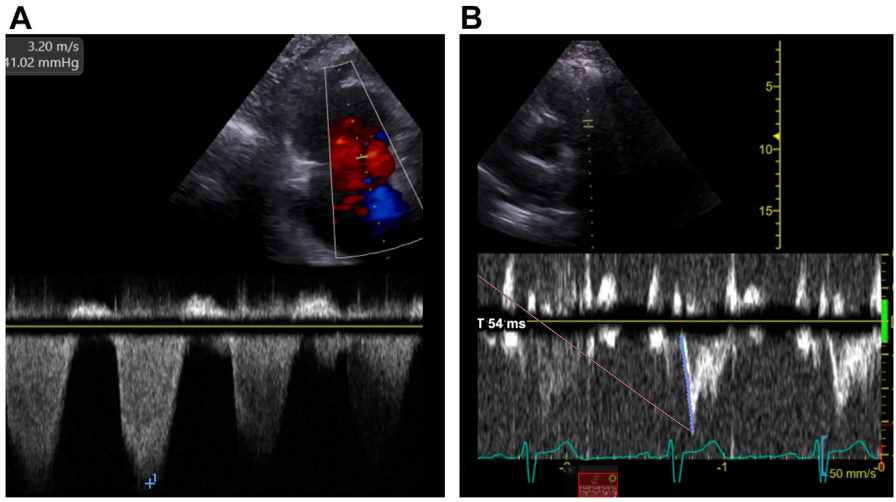

Fig. 8. Shows a 60/60 sign in a patient with intermediate and high-risk PE. Left (*A*) TRPG of 41 mm Hg and right (*B*) pulmonic valve acceleration time of 54 msec.

burden in PE. The more proximal the clot in the pulmonary vasculature, the higher the reflective coefficient and earlier notching, with sensitivity and specificity of 92% and 99% respectively for intermediate and high-risk PE. However, the same study found the ESN sensitivity of only 2% for subsegmental PE.[60]

RV function. RV hypokinesis is a qualitative assessment of RV systolic function with high specificity and low sensitivity in all PE patients, but sensitivity improves significantly when considering only high-risk PE.[54,55] A standardized definition of RV

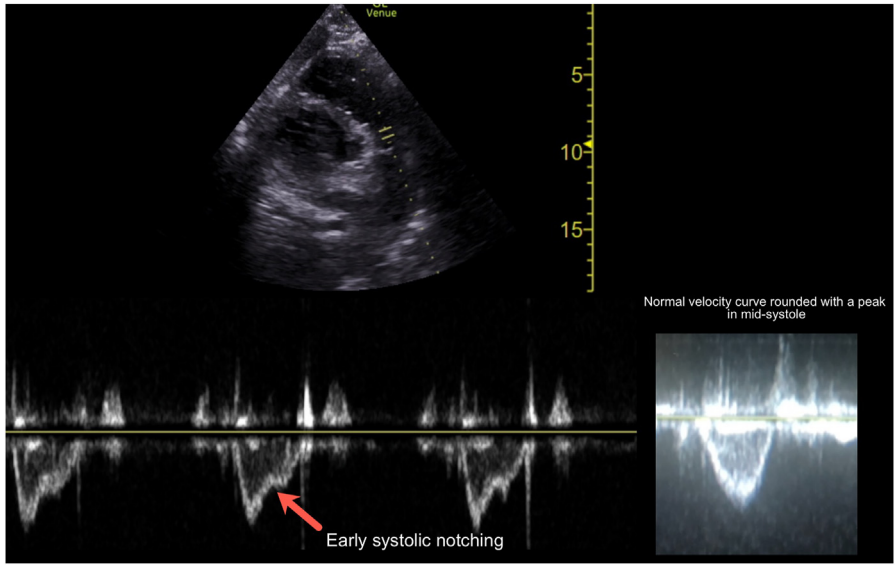

Fig. 9. ESN in a patient with intermediate-high risk pulmonary embolism. The image on the right depicts a normal spectral pulsed-waved doppler at the RVOT.

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dysfunction does not exist, but some studies include RV hypokinesis and RV:LV ratio more than 0.9 as RV dysfunction.[46,61] In early RV dysfunction, the midfree wall is particularly affected, and hence the McConnell sign is distinguished from other causes of RV hypokinesis when apical contraction appears "preserved" or "exaggerated". The McConnell sign was originally reported with a high specificity of 94% for acute PE,[62] with its high specificity confirmed in multiple studies.[53–55,60,63] Despite this reported high specificity, other etiologies of acute and chronic right ventricular dysfunction can present with McConnell sign, including RV infarction, chronic pulmonary hypertension, and acute respiratory distress syndrome.[64,65] The quantitative assessment of RV systolic function by Tricuspid Annular Plane Systolic Excursion (TAPSE) has also been evaluated for the diagnosis of PE. In a study evaluating suspected PE in the emergency department, the sensitivity and specificity of TAPSE less than 2.0 cm were 72% and 66%, respectively.[63] Furthermore, if patients suspected of PE also had tachycardia and hypotension, the sensitivity of TAPSE increased to 94%.[63] Daley and colleagues showed that the most sensitive echocardiographic parameter for diagnosing PE was TAPSE, with a sensitivity of 88% (95% CI = 72% to 97%) for a cutoff more than 2.0 cm, and 67% for a cutoff more than 1.7 cm (95% CI = 72% to 97%). Also, in patients with a heart rate over 110, the sensitivity increased to 93% (95% CI=75% to 99%).[53]

Clot-in-transit. It occurs when an elongated serpiginous clot, that usually originates in the deep veins, gets temporarily stuck in the right atrium or ventricle, but the definition extends to thrombus even seen in superior vena cava (SVC), inferior vena cava (IVC), or transversing a patent foramen ovale.[66] Its prevalence ranges 1% to 4% in low-risk PE, but increases to over 18% in high-risk PE.[46,67,68] Clot-in-transit is highly specific (99%) in diagnosing PE but with very low sensitivity.[55] As shown in Video 1, the concomitant presence of RV dysfunction, along with McConnell sign and clot-in-transit (CIT) is diagnostic of PE in refractory shock.

Multi-organ point-of-care ultrasound

Ultrasound findings in isolation suggestive of a PE have low sensitivity and high specificity, but when lower extremity venous, lung, and cardiac ultrasound are combined, its sensitivity increases significantly. Falster and colleagues, showed a sensitivity of 95% (95 % CI: 76.2–99.9) for multiorgan ultrasound in PE diagnosis.[69] A recent randomized controlled trial showed a 45% reduction in diagnostic imaging referrals.[70] The performance characteristics of single lung, vascular, and echocardiographic findings are included in **Table 2**. The combination of multiple echocardiographic signs improves the performance characteristics of any individual sign,[46] and multi-organ ultrasound outperforms single-organ ultrasound evaluation.[71,72] We propose a multi-organ ultrasound approach along with clinical probability guidance, as seen in **Fig. 10**.

Risk-Stratification and Prognosis of Pulmonary Embolism by Ultrasound

Right ventricle dimension and systolic function assessment

The RV is normally one-third the size of the LV. Both chest CT and echocardiography have been studied to measure the RV/LV ratio, with a ratio greater than 0.9 being highly predictive of increased mortality in PE. In a retrospective study of 950 patients, an LV/RV ratio $\geq$0.9, measured using echocardiography, was independently associated with higher hospital mortality with an odds ratio (OR) of 2.66. Similar results were observed using the RV/LV ratio obtained via CT scan in a systematic review of 49 studies with OR of 2.5 for all-cause mortality 2.5.[73–75]

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Table 2
Structural and morphologic echocardiographic parameters in pulmonary embolism[49,50,107]

Parameter	Views and Technique	Abnormal	Applications in Critical Care	Limitations
RVFW	SC4C, 2D B-mode	>5 mm	RV wall hypertrophy, indicative of chronicity	Perpendicular alignment to RV free wall
IVC	SCIVC, 2D & m-mode	>2.1 cm with <50% collapse with sniff	Reflects obstructive shock physiology if dilated with minimal respiratory variation	Sensitive but nonspecific as observed in mechanical ventilation, hypervolemia, other causes of obstructive shock
RV Basal diameter	RV focused A4C, 2D	>4.1 cm	Indicates dilatation	RV size vary dependent on probe angulation
RV midlevel diameter	RV focused, 2D	>3.5 cm	Indicates dilatation	—
Proximal RVOT	PLAX & PSAX, 2D B-mode	>3.3 cm	Indicates dilatation	Values may differ based on the exact measurement location
Clot in transit	Multiple views	Presence of RA, RV or IVC thrombus	High specificity	Low sensitivity

Abbreviations: PLAX, parasternal long axis; RA, right atrium; RVFW, right ventricular free wall. SC4C, subcostal 4 chamber; SCIVC, subcostal inferior vena cava view.

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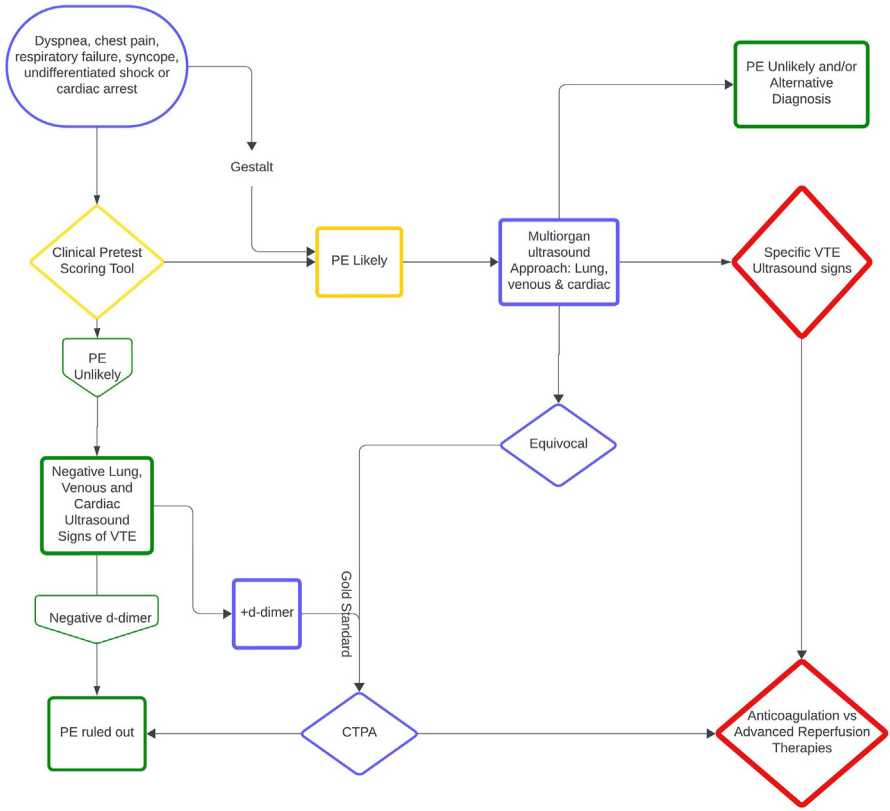

Fig. 10. Proposed ultrasound integrated algorithm in PE diagnosis

A summary of all parameters for systolic functional assessment is included in **Table 3**.

Tricuspid Annular Plane Systolic Excursion. TAPSE measures the displacement of the tricuspid valve annulus using M-mode interrogation in the A4Cview. The m-mode cursor is placed through the lateral tricuspid annulus, and the maximum excursion of the annular plane is measured vertically. The longitudinal fibers of the RV play a greater role in the RV's contractility (80%).[76] For its easy reproducibility, TAPSE is commonly used to assess the systolic function of the RV (**Fig. 7**).[77–80] A low TAPSE has been shown to predict mortality in acute PE.[77] In one study, a TAPSE less than 15 mm was associated with a hazard ratio (HR) of 27.9 for a combined outcome of mortality, and/or hemodynamic deterioration in normotensive PE.[81] Among 450 patients with intermediate-high risk PE, the ratio of age and TAPSE $\geq$4.9 was associated with a HR of 3.24 for 30-day mortality, and 2.02 for clinical deterioration in the next 48 hours, while those with a normal TAPSE had a nearly 100% negative predictive value (NPV) for mortality or need for thrombolysis.[80] Paczynska and colleagues found TAPSE to be better at predicting outcomes compared to RV/LV ratios in normotensive patients with acute PE.[82]

Tricuspid annular peak systolic velocity. Another parameter assessing longitudinal contractility is the measurement of myocardial velocity at the tricuspid valve annulus,

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Table 3
Summary of the right ventricular linear and global contractility parameters in pulmonary embolism[49,50,78,81,105,107,108]

Parameter	Views	Abnormal Values	Application in PE	Advantages	Limitations
FAC	A4C & RV	<35%	RV global function	Evaluates longitudinal and radial function	Errors in tracing
RV Tei Index	A4C & RV	>0.54 TDI >0.43 PWD	RV global function	Less load dependent	Time consuming
TAPSE	A4C	>17 mm	RV longitudinal function	Easy to perform Highly reproducible	Angle dependent
RV S′	A4C	>9.5 cm/s	RV longitudinal function	Easy to perform Highly reproducible	Data paucity
2D Strain	RV Focused	> −20%	RV global function	Reflects contractility of entire RV wall	Data paucity
RVEF 3D	Dedicated multibeat 3D acquisition	<45%	RV global function	Correlates with cardiac MRI RV volumes and EF	Not widely available
Noninvasive RV PV loops	A4C & RV Focused	Ratio E_{es}/E_a 0.5–2.0	RV global function	Correlates with invasive PV loops	Limited to research

Abbreviations: PV, Pressure-volume; RV strain, Right ventricle strain; RV tei index, Right ventricular index of myocardial performance.

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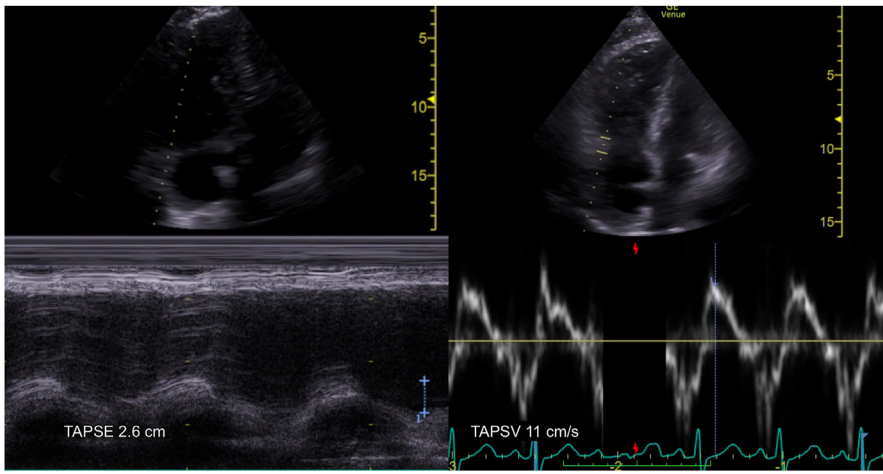

Fig. 11. M-mode on an apical 4-chamber view with the cursor over the tricuspid annulus, measured as the tricuspid annular systolic plane excursion. A normal TAPSE in a patient with intermediate-high risk PE. Tissue Doppler imaging demonstrating preserved RV S′ in pulmonary embolism.

also referred to as RV S'. Tissue Doppler is used to measure RV S′, where the cursor is placed at the lateral tricuspid annulus, and the maximum systolic velocity above the baseline is identified, as seen in **Fig. 11**. An S′ value less than 9.5 cm/sec is an indicator of RV dysfunction. RV S' has limitations in terms of angle dependency, global function assessment, segmental dysfunction, and no measurement of radial contraction. In contrast to TAPSE, it has less robust data as a predictor of mortality in PE.

Right ventricle fractional area change. Right ventricle fractional area change (RV FAC) has been shown to predict the risk of mortality in patients with PE in one small study, but the results were not replicated in others.[50,83,84] One of the major limitations of using RV FAC is the need to obtain an RV-focused apical 4-chamber view with good definition of endocardial borders. Anecdotally, optimal image acquisition for this view is challenging in critically ill patients.

Non-invasive hemodynamics

Measuring hemodynamic parameters of flow and filling pressures aids in risk assessment and prognostication in acute PE. See **Table 4** and **Fig. 12**.

Velocity time integral. The noninvasive calculation of stroke volume (SV), and cardiac output (CO) using velocity time integral (VTI) has risk stratification implications in PE.[85–87] Yuriditsky and colleagues demonstrated that in patients requiring activation of the pulmonary embolism response team, a low LV outflow tract VTI (LVOT) VTI ($\leq$15 cm) was significantly associated with both in-hospital mortality or cardiac arrest (OR 6.0, 95% CI 2–17.9; P=.0014), occurrence of shock and the need for reperfusion therapy (OR 23.3, 95%CI 6.6–82.1; P<.0001). This OR increased to 14 (95% CI 1.7–118.4; P=.015) for the combined outcome of death, cardiac arrest, shock, or requirement for reperfusion in patients with intermediate to high risk PE.[87] Similar results were obtained by Babes and colleagues where LVOT VTI $\leq$ 15 cm was associated with (HR=11.7, 95% CI 4.3–31.6, P<.001), but also found a combined criterion (LVOT VTI $\leq$15 and RV/LV ratio $\geq$1) to have a PPV of 75% and an NPV of 95% in predicting

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Table 4
Noninvasive estimation of right-sided pressures[50]

Estimated Pressure	Formula	Limitations
RAP	IVC <2.1 cm, collapses <50%=15 mm Hg IVC >2.1 cm, collapses >50%=8 mm Hg IVC <2.1 cm, collapses <50%=8 mm Hg IVC 2.1 cm, collapses >50%=3 mm Hg	Gray zone 5 mm Hg–15 mm Hg Limited value in mechanically ventilation Prone to technical errors
PASP=RVSP	4 (TR Vmax)^2+RAP	Requires TR jet
PADP	4 (PR Vmin)^2+RAP	Requires PR
PAMP	79−(0.45*AT) 4 (PR Vmax)^2 +RAP 1/3(PASP)+2/3(PADP)	—
PVR	10 (TR Vmax/RVOT VTI)+0.16	Not reliable in high PVR > 8 WU

Abbreviations: PADP, pulmonary artery diastolic pressure; PAMP, pulmonary artery mean pressure; PR, pulmonic regurgitation; RAP, right atrial pressure; RVSP, right ventricular systolic pressures; TR, tricuspid regurgitation; WU, wood units.

hospital adverse outcomes.[88] Kamran and colleagues used a ratio of pulmonary artery systolic pressure (PASP)/left ventricle stroke volume (LVSV), where LVSV was calculated as product of LVOT VTI and heart rate. They found that PASP/LVSV of $\geq$1.0 mm Hg/mL outperformed traditional PE risk stratification scores in predicting hospital mortality, cardiac arrest, and the need for advanced therapies.[89]

The noncylindrical shape of the RV outflow tract (RVOT) hinders an accurate calculation of RV stroke volume, but the RVOT VTI can be used as a surrogate.[90] The RVOT VTI value and the spectral PWD contour have both, diagnostic and prognostic value. In a small retrospective study, an RVOT VTI less than 9.5 cm correlated with low cardiac output and increased mortality in normotensive PE.[85] Yuriditsky and colleagues also showed that an RVOT VTI less than 9 cm was associated with worse composite outcome of death, cardiac arrest or hemodynamic decompensation in PE.[91] ESN

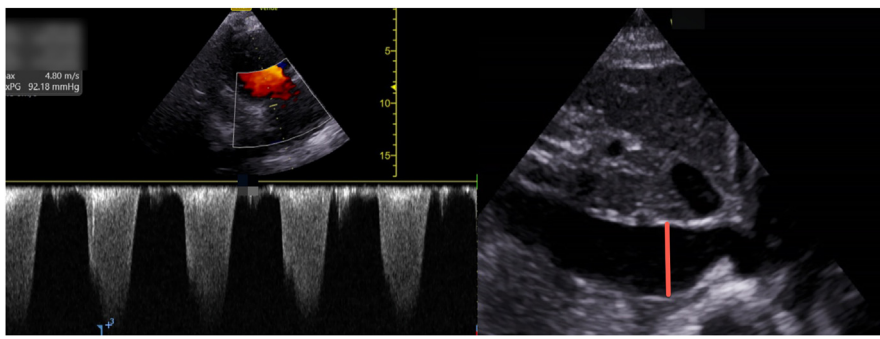

Fig. 12. Once the TR jet (*left*) is identified the continuous wave doppler sample is aligned with the primary axis of the jet. The pressure gradient across the valve is calculated based on the simplified Bernoulli equation. In the absence of pulmonic valve stenosis, the right ventricular systolic pressure is the sum of the RAP and the pressure gradient across the tricuspid valve.

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correlates with severity of thrombus burden, with high sensitivity and specificity for intermediate and high-risk PE.[60] The more proximal the clot is in the pulmonary vasculature, the higher the reflective coefficient and earlier notching.

Clot-in-transit. In a small series, a mortality of over 40% has been reported with CIT, irrespective of treatment.[92] Other cohorts have reported mortality up to 90% in PE with CIT and no intervention.[93,94]

In larger groups of patients, the risk of death from PE is 2 to 5 times higher when there is CIT.[67] A meta-analysis shows that the overall risk of death from PE increases from 4.4% to 16.7% when CIT is present.[95] In a multi-center registry, the presence of PE with CIT, hypotension, and RVD was associated with a 42% vs 12% mortality in PE without CIT. The increased the risk of death is also increased in CIT for normotensive PE with RVD, but not for normotensive PE without RVD. The hemodynamic and RV effects of CIT seem to play a big role in the increased mortality seen in CIT.[96]

Emerging techniques

Non-invasive pressure-volume loops. As previously discussed, RV-PA ventriculoarterial coupling relies on low pulmonary vascular resistance. As afterload increases, RV failure develops once E_{es}/E_a ratio falls below 0.8. The combination of echocardiographic contractility parameters and RVSP/PASP is used to non-invasively estimate RV-PA coupling. In a retrospective study, TAPSE/PASP value of less than 0.4 was identified to have strong predictive value of death or hemodynamic decompensation in PE compared to TAPSE alone.[97] In addition, TAPSE/RVSP predicted a 30-day all-cause mortality, while TAPSE and PASP did not.[97] A post hoc analysis of intermediate-high-risk patients with PE enrolled in the Italian Pulmonary Embolism Registry found TAPSE/PASP $\leq$.33 to be independent predictor of 48-h clinical deterioration (HR 2.06, 95% CI 1.98, 2.11, $P<.0001$) and 30-day mortality (HR 2.28, 95%CI 2.25, 2.33, $P<.001$).[98]

Right ventricle Longitudinal strain. Newer modalities for assessing the longitudinal function of the RV free wall have emerged.[99] Two-dimensional (2D) strain takes advantage of the natural ultrasound "speckles" within the myocardium that are tracked along the RV wall, which makes it less angle-dependent and more representative of global function. Studies have demonstrated that right ventricle longitudinal strain

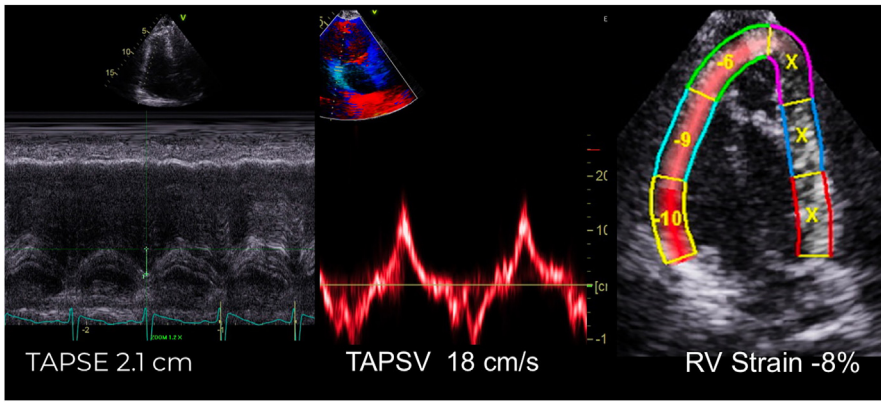

Fig. 13. Preserved longitudinal function by TAPSE and RV S' with reduced apical and free wall strain.

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(RVLS) is a strong predictor of mortality in pulmonary hypertension and acute PE.[84,100–102] Furthermore RVLS correlates with ventriculoarterial coupling in animal models.[103]

The evaluation of RVLS at the bedside is limited logistically by the scarcity of ultrasound systems equipped with these capabilities. Bearing in mind the results of current studies and the prognostic implications of RVLS, we look forward to this modality becoming ubiquitous at the bedside. **Fig. 13** and Video 2 illustrates a case of fatal PE with preserved TAPSE &RV S', but abnormal RV strain.

Three dimensional right ventricular ejection fraction. Unlike the LV, where LVEF is routinely calculated using 2D echocardiography, 2D RVEF volume and ejection fraction assessments are not accurate and not recommended.[49] Three-dimensional (3D) RV ejection fraction (RVEF) is obtained using 3D echocardiography and correlates well with cardiac MRI.[104] Persistent reductions in 3D RVEF on short- and long-term follow-up correlate with unfavorable outcomes in patients with acute PE.[102] A recent study showed 3D RVEF can be used to determine noninvasive PV loops and E_{es}/E_a coupling ratios.[105] This modality is limited to a few state-of-the-art echocardiography departments.

SUMMARY

Ultrasound is pivotal in triage, diagnosis, and risk-stratification of patients with acute PE. Given the significant overlap of signs and symptoms between acute PE and other cardiopulmonary conditions, a comprehensive multi-organ ultrasound assessment can effectively alter the pretest probability for acute PE. Rapid identification of DVT or RVD can profoundly influence the management strategy for patients suspected of having acute PE. Certain echocardiographic findings not only serve as diagnostic marker, but also carry prognostic implications. The current paradigm dichotomizes RV dysfunction as either present or absent, but in intermediate-risk PE, echocardiographic-derived parameters independently add prognostic information. This underscores the potential of echocardiography to predict mortality in acute PE cases, but further trials are needed to confirm this assumption. Anticipated advancements in relatively newer techniques, such as RV longitudinal strain and 3D echocardiography, also hold promise for future integration into bedside assessments in the intensive care unit.

CLINICS CARE POINTS

- Individual signs (eg, DVT, pleural-based lung infarcts, RV dilation) are specific but not sensitive. Combining venous, lung, and cardiac ultrasound improves diagnostic sensitivity to >90% for PE.
- Multiorgan POCUS is especially valuable in unstable patients when CT angiography or other advance diagnostic testing is not feasible.
- V-PA uncoupling, RV dilation and ventricular interdependence lead to reduced LV filling and cardiac output — initiating a fatal downward spiral, even before systemic hypotension occurs.
- RV dilation, RV/LV ratio >0.9, TAPSE <2.0 cm, McConnell sign, and septal flattening are higly indicative of PE severity and associated with higher mortality even in hemodynamically stable PE.
- Clot-in-transit (CIT), though rare, carries a very high mortality risk and requires urgent intervention.

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DISCLOSURE

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SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ccc.2025.02.002.

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